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DISTRIBUTION OF RESIDENTIAL AIR LEAKAGE: IMPLICATIONS FOR HEALTH CONSEQUENCES OF AN OUTDOOR TOXIC RELEASE

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ABSTRACT
Reasonably airtight buildings can protect occupants from large-scale outdoor airborne releases. However, some houses are leaky, as air tightness tends to vary greatly in a housing stock. We modeled the health consequences if a single-family residential community were to "shelter-in-place," for two different models of a toxic release: (I) a simple Gaussian puff, and (II) a realistic simulation of outdoor transport and dispersion generated by the National Atmospheric Release Advisory Center. We predicted the health effects under two different assumptions: (1) every house has the same indoor-outdoor air-exchange rate, or (2) the houses have a lognormal distribution of air-exchange rates. The assumption that every house has the same air-exchange rate (at the median of the actual distribution) can lead to an under-prediction of the community area adversely affected by the release by a factor of 3 or more. The difference is largest if the dose-response relationship of the chemical is highly nonlinear.

INDEX TERMS
Air leakage, air infiltration, air-exchange rate, shelter-in-place, outdoor toxic release

INTRODUCTION
Intentional or accidental large-scale outdoor airborne releases (e.g. terrorist attack or industrial accidents) can cause severe harm to nearby communities. The suddenness of such events means that there is often not enough time to safely evacuate people in the exposed area. Alternatively, taking shelter in buildings can be an effective emergency response strategy. Over 180,000 people were advised to "shelter-in-place" (SIP) as a result of on-site chemical accidents in the US between 1994 and 1999 (Kleindorfer et al. 2003). A recent paper by Mannan and Kilpatrick (2000) listed some successful examples of SIP where injuries and fatalities were prevented.

The effectiveness of simple SIP depends on the ability of building envelopes to restrict the transport of the toxic pollutant to the indoors. There have been a few studies on improving the effectiveness of SIP, such as passive and active filtering (Blewett and Arca 1999), or using duct tape and plastic sheets (Sorensen and Vogt 2001). However, the inherently large variability in the air leakage of the housing stock has not been considered in past SIP modeling. Typical emergency-planning software tools, such as ALOHA (US EPA 2004) and TSIP (Yantosik et al. 2001), predict indoor concentrations using a prescribed or user-defined air leakage constant. The parameter used is rarely tailored to the particular release location, and the health consequences, or predictions of affected areas, ignore the variability in air

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leakage among the housing stock. Predictions of affected areas that include the variability in leakage can differ substantially from those that do not, as we illustrate in this paper.

RESEARCH METHODS

We used two scenarios to investigate the role of the air leakage distribution in affecting the health consequences in a residential community from an outdoor release. The first scenario predicts the outdoor concentration of a toxic chemical with a Gaussian transient puff model, and the air leakage distribution of the housing stock does not vary geographically. The second is a more realistic case in which the outdoor predictions were generated from a detailed atmospheric dispersion model, and the air leakage distribution of the housing stock varies by census tract. In both cases, each house is represented with a one-box model, and the indoor air is assumed to be constantly well mixed.

Case I – Gaussian puff model, fixed air-leakage distribution

We used a Gaussian atmospheric diffusion model developed for short-term releases (Palazzi et al., 1982) to simulate a 1-hour release at constant rate, with a constant 5 m/s wind. The source is assumed to be non-reactive and perfectly reflected at the ground. Dispersion coefficients were chosen to correspond to standard Pasquill-Gifford stability class C, representing a slightly unstable atmosphere.

The model grid has a resolution of 5×5 m. There is no attempt to represent individual houses; instead, at each location we create a statistical distribution of houses. We assumed a normalized leakage (NL) distribution with geometric mean (GM) = 0.5 and geometric standard deviation (GSD) = 2.0. This distribution is based on earlier analysis of 70,000 blower door measurements taken in US houses (Chan et al. 2003). We use the LBL Infiltration Model (Sherman and Grimsrud 1980) to predict the resulting air-exchange rate distribution, as assuming closed-house conditions. Assuming a 10 ^{o}C indoor-outdoor temperature difference and 5 m/s wind, the pressure difference on the building envelope will lead to a lognormal air-exchange rate distribution with GM = 0.61 h^{-1} and GSD=2.0.

Acute inhalation toxicity studies show that many chemicals exhibit nonlinear dose-response relationships (ten Berge et al. 1986): For chemicals such as ammonia and chlorine (n>1), exposure to an extremely high concentration for a short time can be much more dangerous than exposure to lower concentration for a proportionally longer time. Health responses at time *T* can be reasonably predicted from the toxic load $L(T) \equiv \int_{t=0}^{T} [C(t)]^n dt$, where C(t) is the concentration as a function of time, and n is a constant known as the toxic load exponent (which need not be an integer). For chemicals with n = 1, the toxic load is just the time-integrated concentration, or "exposure." The acute exposure guideline levels (AEGLs) recently established by the National Research Council (NRC 2000) on many toxic industrial chemicals and warfare agents use the toxic load model to predict health outcomes.

We simulated a release of a fixed amount of material and selected three toxic load exponents (n = 1, 2, and 4) when analyzing the heath consequences. The range of n is chosen to represent typical values observed in toxicity studies. At each grid cell, we determined whether house occupants would exceed a given toxic load level (TLL); that is, whether *L(T)* > TLL. The TLL for the case of n = 1 (TLL_1) is chosen such that the release material has roughly the same toxicity as chlorine gas. TLL_2 and TLL_4 are then scaled according to TLL_1 such that the total number of people exceeding TLL, if people were uniformly distributed *outdoors*, is the same in all three simulations.

Case II – Atmospheric dispersion model, spatially varying air-leakage distribution
For a more realistic simulation of an outdoor release, we used the output of a 3-D atmospheric dispersion model from the National Atmospheric Release Advisory Center (NARAC). The hypothetical release lasted for 1 hour. The model grid is 48×48 km and centered near downtown Albuquerque, New Mexico. The simulation used actual meteorological input data from February 24, 2003 from 18:00 to 22:00.

The normalized leakage distribution of a housing stock can be predicted from information about the age, floor area, and household income for the houses in the area (Chan et al. 2003). We used data from the US Census and the American Housing Survey to predict the statistical distribution of normalized leakage for each census tract in Albuquerque. The LBL Infiltration Model was then used to predict the distribution of air-exchange rates at each grid point. Unlike in Case I, each grid point has a unique distribution of air-exchange rates, which depends on both the census tract the grid point is located in, and the time-varying meteorological conditions at that location.

For a given distribution of air-exchange rates, the method used to predict indoor concentrations and health consequences is the same as in Case I. In both cases we ignored pollutant dynamics, such as sorption to indoor surfaces or penetration losses to the building envelope. Three toxic industrial chemicals are used to illustrate the effect of different toxic load exponent on the predictions: methyl isocyanate (MIC, which has $n = 1$), chlorine (Cl_2, $n = 2$), and hydrogen sulfide (H_2S, $n = 4.4$).

RESULTS AND DISCUSSION
Figure 1 shows (left column) the predicted number of people who will exceed the TLL, as a function of time from the onset of the release, for Case I. The dashed line represents the more realistic case of houses having a lognormal distribution of air leakage, while the solid line shows the simplified case of all houses having the same air leakage equal to the median (GM) of the lognormal distribution. For all toxic load exponents n, the number of people exceeding the TLL is found to be about the same, at all times. However, for $n > 1$, even though the total *number* of people affected is about the same under the two assumed leakage distributions, the *locations* of the affected people are predicted to differ.

The predicted areas shown in Figure 1 are evaluated long after the release has ended, so they represent the eventual affected area owing to the release. In presenting our results, we define the "affected area" as all grid points for which more than 5% of houses exceed the TLL. The different colors (right column) represent the predicted percentage of houses in the area that exceeded the TLL. Ten percentile ranges are displayed: > 95% people exceeded the TLL, > 85%, ..., and > 5%. The maps are identical for $n = 1$, since long-term exposure is the same for all houses regardless of their air-exchange rate as long as the air-exchange rate does not change with time. For $n > 1$, however, the affected area is about twice as large for the realistic case as if all houses are assumed to be identical. In the realistic case, even a long way from the release source, people in the leakiest houses are at risk.

In the more realistic simulation of a hypothetical release in Albuquerque (Figure 2), the predicted number of people exceeding the TLL (left column) again depends only on the median leakage and is not sensitive to the variability of the leakage distribution. For the constant-NL runs, we used a normalized leakage equal to the population-weighted GM of the 11 census tracts within a 6.8×4.3 km boundary around the release location. Compared to Case I, the release created a less uniform plume covering more area because of changing wind direction. Figure 2 shows the "affected area" 3 hours after the end of the release. By this time, people should have left their homes (or opened their windows) and thus stopped accumulating toxic load.

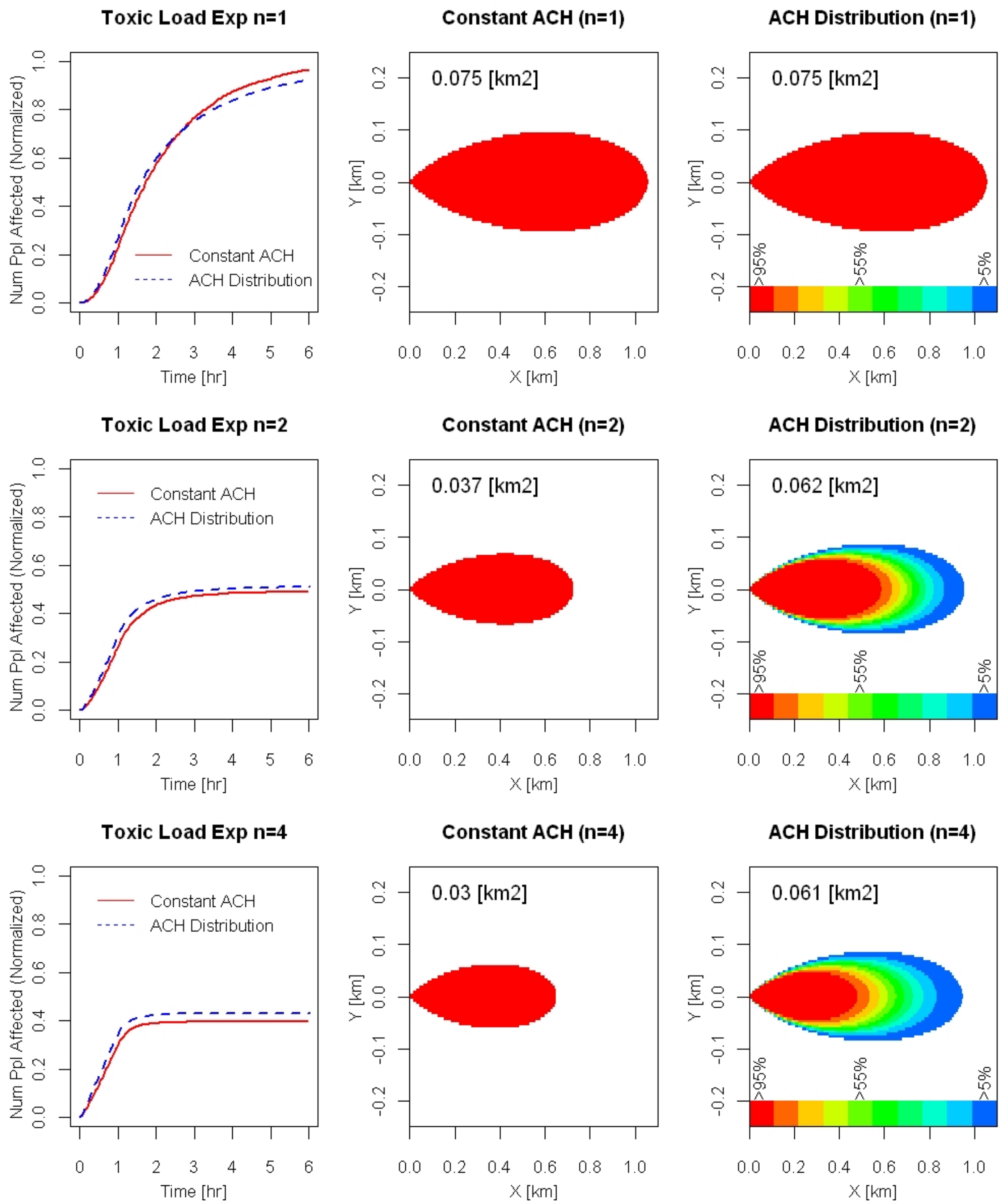

Figure 1. *Gaussian plume simulation results (Case I): (left column) predicted number of people exceeding the TLL if everyone were indoors, divided by the eventual number if everyone were outdoors; (center column) predicted "affected area" if all houses have the same air-exchange rate (0.6 h^{-1}); (right column) affected area if air-exchange rates are lognormally distributed (GM=0.6 h^{-1}, GSD = 2). The total affected area in km^2, indicated at the upper left of each area plot, includes all colored areas shown.*

For all three chemicals (MIC, Cl_2, and H_2S), the affected area is larger when the variability of house leakage is included. Unlike Case I, this is true even for n = 1 (MIC, shown in the first row). Terminating the accumulation of toxic load at $T = 4$ h "freezes" the health effect maps, which would otherwise continue to change (the constant-NL map would grow slightly, while the NL-distribution map would stay about the same size but shift more towards the red). The same is true for n > 1 (Cl_2 and H_2S) but to a much lesser extent. This is because with a higher toxic load exponent, the dose leading to an ultimate health burden is most highly concentrated during the early phases of the release when the indoor concentrations are the highest. As the plume passes, the low level of residuals left indoors contribute much less to the overall toxicity resulting from the exposure.

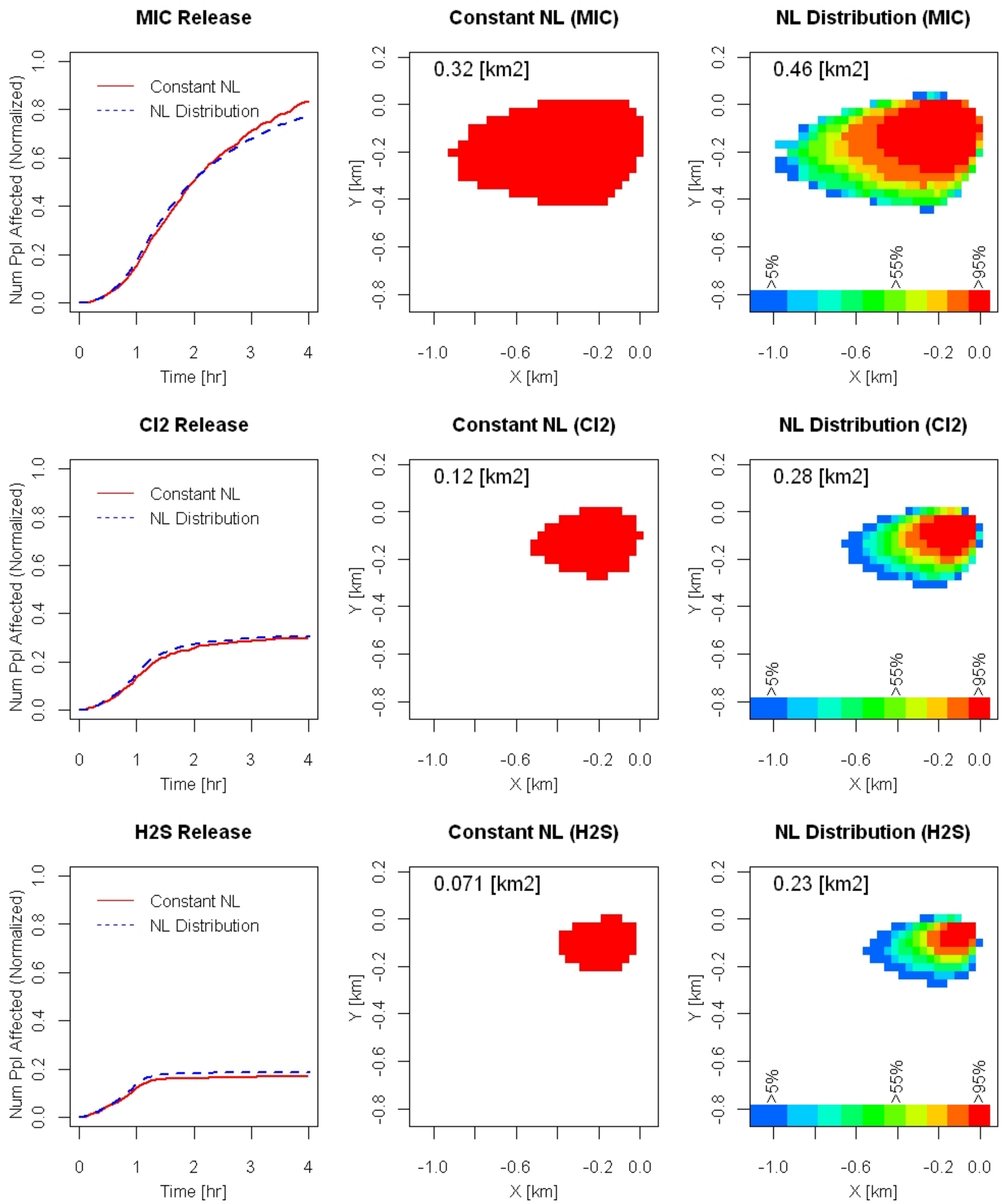

Figure 2. *Hypothetical release in Albuquerque (Case II): (left column) predicted number of people exceeding the TLL if everyone were indoors, divided by the number at four hours if everyone were outdoors; (center column) "affected area" if all houses have NL = 0.7; (right column) affected area if leakage variability within each census tract is included. All affected areas are evaluated 4 hours after the start of the 1-hour release.*

CONCLUSION

The air leakage distribution of houses influences the area affected by an outdoor release, when people take shelter in houses. Some people may receive a dose that exceeds the TLL because they are sheltering in leaky houses, even though they are far downwind of the release, and this will not be correctly modeled if the variability in house leakiness is ignored. The effect of this error is largest when shelter-in-place is terminated soon after the outdoor plume passes, and when the toxic load exponent is large.

Health effects from an outdoor toxic release will also be affected by many factors not considered in this paper. For example, certain chemicals sorb significantly onto surfaces and some chemically decompose or otherwise react with compounds in the environment. Furthermore, although we have assumed that each house can be represented as a single well-mixed box, in reality the concentrations in a house may vary from room to room. Including these factors would change the details, but the basic conclusion appears robust: ignoring the variability of air-exchange rates among houses in the housing stock will lead to substantial misprediction of where casualties are likely to occur.

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